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WITH ABSTRACT OF THE LITERATURE

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Linseed Dermatitis

Employment "Sickness and Death Rates"

Studies on Experimental Pneumonokoniosis. VI. Inhalation of Asbestos Dust: Its Effect upon Primary Tuberculous Infection

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THE JOURNAL OF INDUSTRIAL HYGIENE

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LINSEED DERMATITIS*

M. H. BARNES, M.D.

THE linseed oil crushers, manufacturing linseed oil, have an abnormal labor turnover, owing to the fact that some of the workers contract a linseed dermatitis which in many instances makes it necessary for them to obtain other jobs. For a correct understanding of the way in which the dermatitis is contracted, a brief account of the source of the material and the process of manufacture is necessary.

TYPES OF FLAXSEED USED

The linseed oil crushers in this country draw their requirements from four different sources: namely, the United States, Canada, Argentina—which together supply 90 per cent. or more of the seed—and India. The chief difference in the various seeds is in the oil content. The American seed runs 18 pounds of oil to the bushel, the Argentine seed 19 pounds, and the Canadian 20 pounds. For practical purposes the Argentine seed produces as good oil as the others, and is not of inferior quality as it was formerly considered.

PROCESS OF MANUFACTURE

The methods of manufacture in the various mills are essentially the same. For one large mill the process is as follows: The flaxseed is unloaded by means of a series of conveyors and finally reaches a cleaning bin. Here the dust is removed by a suction fan, and the seed passes over various sieves which remove wheat, oat, mustard, and grass seed, together with other impurities. It is then put in a large bin on the top floor of the mill, and from here it passes through crushing rolls which reduce it to a very fine meal. This meal is placed in steam jacketed cookers and is dropped from one section or deck of the heater to the next by means of an agitator inside the cooker. When it finally reaches

* Prepared under the Industrial Department of the DeLamar Institute of Public Health of the College of Physicians and Surgeons, Columbia University, New York City.

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the lowest stage, it is up to a temperature of 200°F. It is then withdrawn into a mold and made into a cake. The cake is wrapped with cloth made of human hair which comes from China, and is placed in a hydraulic press where most of the oil is extracted. The oil is taken to the refinery for refinery purposes, while the cake is packed and sold as a by-product.

Review of Literature

From the available literature on the subject, the following description of the dermatitis can be formulated.

Definition.—Linseed dermatitis appears as maculopapular lesions, showing frequent red follicular papules. It occurs chiefly on the dorsal surface of the hands, and on the anterior aspects of the forearms and thighs, but occasionally on the face, upper arms, and feet. The rash produces a severe itching or burning sensation.

Incidence.—The incidence varies with the source of the seed and the season of the year. In making a survey of the operations of one company, the following facts were obtained: In mill No. 1, where United States and Canadian seed is used exclusively, the workers of Scandinavian or Polish origin are most susceptible. In mill No. 2, where the seed used is exclusively from Argentina, and where the pressroom laborers are chiefly negroes, they have less trouble than in mill No. 1. Occasionally they employ a Polish or Scandinavian immigrant, who is extremely sensitive and develops a severe rash which necessitates his leaving the mill. The inference, then, is that the negro is less susceptible and that the source of the seed has nothing to do with the development of the rash. At mill No. 2 the rash occasionally occurs among the men working around the screen and storage bins, but it is more frequent among the pressroom workers. The incidence is highest during the summer months.

From inquiry into the procedure of another company, the following information was secured: The rash is not serious. Practically everyone that works in the pressroom contracts it sometime during his work there. It is caused by the linseed oil's being continually on the arms and hands, and in order to overcome this the workers are taken away from the pressroom for a period of a week or so, during which time the rash disappears. Everyone is susceptible to it, some more than others, owing almost entirely to the general physical condition.

Etiology.—While the etiology is unknown, the suggestions have been made that it is a dermatitis venenata due to an impurity in the seed, and that the seed from India and Argentina is more irritating than the seed from the United States and Canada.

Pathology.—Macroscopically the lesions are symmetrical, discrete, and quite deep. They are located as a rule on the dorsal surface of the hands, and on the anterior aspects of the forearms and thighs; occasionally they appear on the neck, chest, arms, and feet. The rash generally shows thickly scattered macules and papules with frequent red follicular papules associated with secondary scratch marks and crust formation. There is no record of any microscopic examination.

Symptoms.—The symptoms vary with the length of exposure, the season of the year, and the susceptibility of

the person. As a rule the chief complaint is an itching rash on the arms and thighs. One man had an eruption, with itching, on the anterior surfaces of both feet. The itching is worse at night and after bathing. Some patients state that they can get relief only by scratching the lesions until they bleed. In other patients, the most marked symptom is a burning sensation accompanied by some itching. The lesions have also been described as a weeping eczema with itching and burning.

Differential Diagnosis.—The lesions of linseed dermatitis must be differentiated from macular or papular syphiloderm, papular eczema, and scabies. The syphilis rash may be easily eliminated by an accurate history and serologic tests; without these aids the differentiation is more difficult. The macular syphiloderm appears as discrete macules of pea size or larger, of a faint pink or red color. The eruption is, as a rule, more generally distributed over the body and is more pronounced over the chest and abdomen. There may, of course, be evidences of chancre, with enlargement of the glands with or without appreciable systemic and subjective symptoms. The Wassermann and Kahn reactions may be positive. The small papular syphiloderm is seated around the hair follicles, and consists of firm pointed papules, widely distributed and especially abundant on the trunk and chest; there is no itching or exudation.

In papular eczema, the papules are discrete or closely crowded, round or acuminate, and of pin point to pinhead size, and are frequently topped by vesicles. Excoriations and blood crusts produced by scratching are evidence of the intensive itching. Papular eczema may be very difficult to differentiate from the linseed dermatitis. The history of working in the pressroom or around the bins of a linseed oil mill and the disappearance of the rash after staying away from work, or by changing jobs, may be the only means of differentiation.

At times, scabies resembles the linseed itch very closely. It should, however, be easily distinguished by the history, the multiform lesions, the cuniculus, and frequently by the location of the lesions.

Treatment.—The treatment consists chiefly in removing the worker from the storage bins and pressrooms to some other job where he will not be further exposed to the flaxseed or linseed cake. As a rule, if he stops work for three or four days, the irritation immediately disappears.

In most cases the irritation has been accelerated by the use of burlap to wipe the oil and meal off the hands and forearms between pressings; this material in itself is exceedingly rough and irritating and is more or less contaminated with infectious substances. The use of sterilized rags and towels, together with a thorough cleansing with soap and water after the work period, has been effective in reducing the incidence and the severity of the linseed dermatitis.

In the more aggravated cases, a week's rest is required, with careful cleansing of the lesions with soap and dressing with a sulphur salve combined with 16 per cent. balsam of Peru.

Prognosis.—As a rule the dermatitis is not of a serious nature, and removal of the patient from the exposure of his

work, together with simple cleansing of the skin lesions with soap and water, and dressing with sulphur ointment, is all that is required. If the patient continues to work in the same part of the mill, while suffering from the dermatitis, more severe reactions may occur as a result of infection.

Three of the articles reviewed have no direct bearing on linseed dermatitis, but they may, indirectly, throw some light on the problem:

Dr. Smith (7) states that he was called to see a boy who was suffering with severe nausea and vomiting, swelling and redness, and marked irritation of the tongue and fauces. The examination and history suggested some kind of poison taken by mouth. The boy was given a strong cathartic, followed by fluids, and he had practically recovered on the following day. On recovery he gave a history of having eaten a piece of linseed cake.

Black (8) states a very similar clinical observation. In this case the linseed was a component of a breakfast food, and the patient was an adult. He experienced swelling, tingling, and burning sensations of first the mouth, then the pharynx and esophagus, from eating a small amount of the breakfast food. The conclusions drawn in this observation are that flaxseed apparently causes more violent local and general reactions in the hypersensitive than do most of the other allergic food substances. In this case there was a history of contact with flaxseed in the form of poultices, which might have served to sensitize the skin.

A third article (9), which may be still more remote from the subject, gives the following information. In one of the eastern fish hatcheries, linseed meal was purchased for the purpose of economy, as it was much cheaper than the food formerly used in feeding the trout. The fish reacted by turning dark, floating on their sides, and finally dying. On analysis it was found that the linseed cake contained a glucoside which on hydrolysis produced a small amount of hydrocyanic acid, which caused the death of the fish.

Experimental Study

The object of this paper is to collect and correlate the literature on linseed dermatitis or linseed oil dermatitis and perhaps, by a few simple experiments, to pave the way for a more extensive study of the etiology, methods of prevention and, if possible, of desensitization of workers suffering from the dermatitis.

Observers have considered that the rash is due to various causes: (1) a blockage with resulting inflammation of the sebaceous glands caused by the oil's being in contact with the skin for eight or more hours a day; (2) an allergic reaction or sensitization to burlap, which is used by some workers to wipe the oil off the parts of the body exposed; and (3) a type of dermatitis venenata due to impurities in the cake or the seed, in which an individual idiosyncrasy or sensitivity plays an important rôle.

Seed and samples of linseed cake were obtained from two large companies, and were used to make various preparations for test purposes:

1. The impure seed was ground up to a meal, mixed with a small amount of distilled water, and heated in a water bath to approximately 200°F.

2. The impurities from the Argentine, Canadian, and United States seed were

mixed together and ground up, a little distilled water was added, and this mixture was heated in a water bath to 200°F. Examination by means of a hand lens showed that the impurities from the three different seeds were essentially the same. As far as could be determined, they consisted in wheat, oat, and barley seeds, wild grass seed, and mustard seed, together with small flecks of chaff and pieces of grass, leaves, etc.

3. The pure seed was ground up, mixed with a little distilled water, and heated in a water bath to 200°F.

4. The linseed cake was ground up and heated together with a little distilled water to a temperature of 200°F.

5. A bottle of pure linseed oil was the fifth preparation.

Twenty patients attending the Industrial Clinic consented to be subjects of the experiment. Five patch tests, one for each preparation, were applied to each person. With the patch containing linseed cake paste, two of the twenty showed a definite maculopapular eruption, accompanied by severe itching, and several others complained of itching and burning sensations but showed no definite eruption. One showed a maculopapular rash, with itching, from the patch containing the impurities. There were one or two doubtful reactions produced by the impure and the pure linseed patches; the most that could be detected was a mild macular rash accompanied with some itching. In no case did the linseed oil patch produce a dermatitis.

It is difficult to simulate the condition of exposure in the linseed oil mill by means of patch tests. If the linseed cake patch could have been kept hot and moist for a period of eight hours a day, and some irritation could have been applied by rubbing the patch over the skin area, more reactions would doubtless have been observed. The patches were kept on for a period of one to three days, the length of time varying with the patient's wishes and with the requirements of the clinic.

In one of the patients a biopsy was performed, and while the microscopic picture is not diagnostic in itself, it was considered worth reporting:

MICROSCOPIC DIAGNOSIS: Acute Dermatitis (Linseed Cake Dermatitis). C. M.—O. P. D. 178302—Biopsy 4147.

This section shows a marked edema of the epithelium. Here and there a few cells infiltrate the epithelium. The corium also shows a marked edema and an increase in nuclei which are apparently those of round cells, chiefly of a lymphocytic type. There is also a rather marked infiltration about a few of the more superficial vessels of the corium, but similar types of cells are seen. A few cells are definitely polygonal in shape and an occasional polymorphonuclear leukocyte is observed.

As most of the men who develop linseed dermatitis work in the press-rooms where they handle the hot, moist cake from the steam jacketed cooker, it was considered probable that the irritating substance in the cake was water soluble, and a water extract was prepared as follows:

Four to five ounces of ground-up linseed cake were mixed with a small amount of distilled water. The procedure was similar to the preparation of thin breakfast food. This mixture was heated for three hours in a water bath at a temperature of about 200°F., and was then filtered. Approximately one ounce of filtrate was obtained, which was opalescent and resembled diluted skimmed milk. This filtrate was then sterilized in a steam autoclave for two hours.

Ten patients were given intradermal injections of the water extract, normal saline being used as a control. The technic was the same as the Mantoux

test, except that 0.1 to 0.2 c.c. of the extract was used for each injection. Of the ten patients, four showed positive reactions, two showed mild reactions, and four were negative to the test.

One of those who gave a positive reaction was the patient on whom a biopsy was done for a positive linseed cake patch test. This patient showed a delayed positive reaction, the reaction to the intradermal injection of linseed cake water extract reaching its maximum twenty-four hours after the injection. There was definite redness and erythema in an oval area, 9 cm. by 7 cm., surrounding the point of injection.

Two other patients reacted similarly. The author was the fourth patient. Up to the age of 14 years he had bronchial asthma, and at present is susceptible to mild attacks of hay fever and moderately susceptible to ragweed and horse dander. The injection of 0.1 c.c. of the solution intradermally caused marked burning and itching, while the normal saline intradermal injection caused no reaction. This reaction on injection is similar to the symptoms described by each of the other patients. The injection was done at 3.30 P.M. At 4 P.M. there were all the symptoms of hay fever, red and watery eyes and a marked discharge from the nose, with a general uncomfortable and irritable feeling. At the site of injection there was a raised red area of skin, measuring about 2 cm. by 3 cm., and a surrounding much larger zone of erythema. At about 4.15 P.M., the attack resembled a severe attack of bronchial asthma with hay fever symptoms superimposed. The symptoms were as follows: rapid pulse, difficult breathing, using the accessory muscles while leaning on the window sill of an open window; watery discharge from the nose, eyes red and watery, profuse sweating; frequent coughing, raising a frothy thin mucus but no mucous pearls; face red and congested; lips felt swollen, and tingled; irritable, depressed, and with a feeling of beginning suffocation. At about 4.45 P.M. the symptoms were the same as those given above, but they had diminished to about one-half of their maximum intensity. In addition, there was marked itching and burning around the neck and on the body and thighs. The whole volar surface of the forearm was raised, red, and somewhat tender, imparting a mild burning sensation. Extending up the arms were chains of large maculopapules, which were red, and tingled or burned slightly. Over the body of the opposite arm, trunk, and thighs there were numerous faint red macules, varying from $\frac{1}{4}$ cm. to $\frac{1}{2}$ cm. in circumference.

The symptoms gradually diminished until 8.30 P.M., when the rash and maculopapules had practically faded out, though the volar surface of the forearm still showed about 20 per cent. of its maximum reaction.

Conclusions

While the results from the brief series of cases reported in this paper are far from being conclusive, they indicate that linseed cake contains a skin irritant to which some individuals react without previous sensitization and that this irritant is not present in pure linseed oil or in impurities removed from linseed by passing it through sieves.

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EMPLOYMENT "SICKNESS AND DEATH RATES"*

HAROLD W. STEVENS, M.D.

THIS paper does not propose another explanation or another remedy for unemployment. It aims rather to emphasize a concealed and little-understood factor constantly involved in unemployment, casting a shadow of fallacy over explanations which ignore its presence and a shadow of futility over proposed remedies which fail to take account of its influence.

Is labor a homogeneous commodity measurable in standard units transferable and adaptable to new needs? Is unemployed labor a surplus comparable with surplus capital and surplus products? Can the creation of new industry and improvement in the distribution of labor; can shorter hours, fewer days per week, or any one or all of the mechanistic manipulations of industrial and financial management supply the real needs of the unemployed? Or is unemployment an inevitable hazard of civilization to be dealt with by insurance coverage or other device for social distribution of its burden?

The daily, routine activities of employment management, welfare management, medical supervision, and other business functions dealing with the intimate facts of unemployment in the making are teeming with information pertinent to these unanswered questions.

* Received for publication Nov. 17, 1930.

An old man appeared at the door of my office in the dispensary. With wide, anxious eyes and with tears dropping down his cheeks he cried out: "They let me go; they let me go. He say: 'Got to lay you off; bisness gone down flat.' Doctor, can you do someting?" The man was about 60 years old; he had worked for the business twelve or fifteen years—a war time replacement, I believe. He was not disabled though he had infirmities amounting to some degree of handicap. He was one that must inevitably have been marked as a prospective liability for the business. He would certainly find it difficult or impossible to get other occupation. I knew there was nothing in the facts very likely to turn the tide of "turnover" in his favor; but I could only answer, though with a twinge of self-reproach for my unwarranted optimism, that I would see what I could do. The man had been my patient; and so, waiving the usual etiquette, I inquired of the employment division about the circumstances of his discharge. The reply was: "Left without notice."

I knew of no way of verifying these apparently conflicting stories. There was at the time no conspicuous indication that the business had gone down flat financially; though in its sense of social responsibility it may seem to have gone a bit flat. There appeared not the slightest cause to believe that the man had left voluntarily, as the final employment record seemed to indicate.

A young woman of 26 or 28 came to me one day with the anxious entreaty: "Do let me come back. He said he knows positively that I'm well, but I'm not very anxious to help myself; and then you doctors won't let me come back; so of course they can't help me any more." (The "He" referred to was the welfare manager.)

This girl had been absent from her job in an embroidery workroom for seven years,

after having worked only three or four years with the business. During this absence she had been observed and investigated by numerous physicians and hospital clinics. Along with other ailments the diagnosis of pulmonary tuberculosis had been made; and during the entire time she had never been free from some indication that the disease was still active, although she gained weight and acquired some appearance of health. Her social situation as an orphan was unfortunate, but an exceedingly generous personnel management of the business had continued to aid her with medical care and financial support amounting to several thousand dollars, though there had never been any recognized industrial cause for her disability. For some time, however, it had been growing evident to me as medical adviser that it would never be fair to the girl to approve her return even on much limited work. Furthermore, I had been assured by high authority in the management: "We don't want her back—we wouldn't take her back."

But now she "just must come back, because they won't help me any more, and I got to live." With flushed cheeks and glittering eyes she insisted: "Now I *am* willing to help myself and I want to come back, because he says he knows positively that I'm well and able to work." I am certain that my reasoning was vain to make her see that her return to her former job under existing conditions would be a foolish move for everybody concerned.

Fortunately for this girl she had found a home with a church institution during the later years of her disability; but now the question arose: "What will happen with all income cut off?" Within a few days I found opportunity for a talk with the sisters of the institution. I was answered that, income or no income, the church would find some way to take care of the girl; but their comment upon the attitude of the business as represented to them by its welfare manager was this: "We agree that the business *has* done a great deal for this girl. We do not agree that she is well; and we know that she is not able to work."

Somewhat over a year after this episode I inquired again in regard to the girl's progress. She was still being cared for by the institution. She still had afternoon temperatures, and was under medical supervision and nursing care; and the sum of her economic productivity was represented by one or two hours of embroidering daily.

There are several obvious comments upon this story. First, the generous compensation of this girl's disability for a period of several years far exceeds anything that could reasonably be expected from any business. Also, any reasonable person with some practical health knowledge would agree that a girl in such a condition might with benefit to herself engage in some limited and supervised occupation. Finally, the position taken by the business, in at last dismissing this girl with the announced opinion that she was able but unwilling to work, utterly misrepresented her true relation with the community.

Thus every day adds its contingent of dismissed workers who pass out from mine and factory, store and office to mingle their peculiar handicaps with the great mass of undifferentiated misfortune, which is unemployment. It is true that in many employment discharges or other "separations" the important individual characteristics are not so conspicuous or so easily identified as in these stories just related. Gross disease or defect might not be found in any large proportion. I am, however, certain from my own observations and from the opinions of other observers that these instances related (neither of which occurred in 1921 or subsequent to October, 1929) are not exceptions or rare examples of emergency; I am convinced that in a large proportion, if not all, employment discharge transactions there may be identified a vital human element which is officially buried alive, with the only marker some ambiguous classification symbol which suggests that employment rejection is a most impersonal and circumstantial matter; while

on the side of the dismissed employee the human truth becomes merged in the inarticulate murmurings of disappointment and discontent which intermittently rise to audible complaints and stir our charitable or socialistic impulses and resolves.[1]

The socially minded student of ideal human relations is stirred by the fine potentialities of business as an instrument for better things and for more discriminating understanding and good will among men. He may be shocked by his first acquaintance with some of the conventional equivocations of business in dealing with its personnel. It is not, however, my wish to make out a case for abused laborers against unjust employers. I would not for a moment suggest that there is anything reprehensible in the practical adjustments which the demands of progressive business may require. I have no thought of complaining about the essential principles of business. The principles of business, when judged by its finest exponents, are not excelled by those of any calling—excepting none; and the principles of business in the gross are being surely and justly tried by practical social judgments quite apart from and unaffected by any individual opinion derived from the ethics of personal relations. I know what it is to try to help distressed, aged, or otherwise disabled workers to face an uncertain future, unemployed. I know also what it is to face with a perplexed employer the increasing burden imposed by none too discriminating popular sentiment concerning the social obligations of industry to its employees. Knowing something of both questions, it seems to me that they must be regarded as equally perplexing elements of the greater social problem. My real purpose is to appeal for a more straightforward and business-like dealing with these discharge transactions, and for a more rational and common sense use of the facts derived therefrom—facts to which we have not yet had the vision and the courage frankly to adjust our industrial relations.

Unemployment viewed from the vantage point of a personnel executive, but with natural vision uncolored by the protective screening often developed as a shield against inevitable soul disturbing misgivings, is the product, or by-product, of a relentless process of selection and rejection. The process by which one is taken and the other left is not a process of mere chance, nor is it determined by impersonal and circumstantial exigencies of business. The individual case of unemployment is usually the result of some personal decision, oftentimes after much study and deliberation, that one individual will perform better than another under some set of rather personal standards called a job. Opinions regarding present and future health, appearance, character, work performance, race, sex characteristics, age, and even social and family connections and personal grudges enter into these important decisions. In fact, it seems probable that only in the relatively rare instances of the complete dissolution and disbanding of a business is employment discharge entirely independent of this discrimination;

[1] The Adult Hygiene Division of the Massachusetts State Department of Public Health is at present engaged in certain studies, soon to be published, which should yield some of the first systematized information upon these matters.

even then, in the process of adjustment to new employment the worker discharged under such conditions soon finds himself in some other industry's long waiting line from which one in many is picked for assignment to a job.

Workmen's compensation and other recent industrial legislation have fixed attention on physical fitness as a precaution against legal liability for the employer. Health and welfare services for industrial workers have emphasized the physical and mental factors of fitness and disability. Even so remote a factor as the persistent demand for lower and lower prices, in which we all participate, is passed on to become a part of the demand for efficient industrial performance. All these influences have added to the discounting of inefficiency and have thus contributed to the present magnitude of the enigma of unemployment.

It is no dramatic revelation that in a certain important respect unemployment is the result of a process of selection. It is somewhat surprising that we should cling so long to the illusion that all responsibility in these intensely personal issues may be discharged by ignoring them, or by passing them off upon impersonal "business conditions." Every discharge or refusal to employ means that some one has been weighed in the balance of employment standards and found in some respect wanting. Yet in many, or most, instances of employment discharge, neither the individual nor the community ever knows the real reason. The dismissed employee is sent out to try his fortune elsewhere, his only endowment bestowed by his former employer a possible money allowance and the onus, or the honor, of having been discharged. The answer to his eager questioning is "the condition of business" or that even more mysterious thing, "policy"; when he might be discreetly admitted to the great secret that even the condition of business and policy have some rather personal elements; or at least he might be tactfully and helpfully told that the reason for his discharge is something specific and concretely personal about which he himself might do something intelligent and constructive in the way of remedy, or in the way of finding himself industrially. Unemployment is to the individual an exceedingly personal matter; can we hope by social measures to provide genuine help until we can incorporate that personal element into our social concept of unemployment?

It is easy to believe that if we understood these matters better, much of the mystery about employment relations would be dispelled and new light might come to other vexing issues of our social economy. More professional discrimination and integrity in employment methods would add refinement in the quality of the judgments that assign individuals to the class of the unemployed. Physical and mental rating for industrial purposes would be seen to have only begun its real task. The persistent obsession that all men must be dealt with as if we believe them equal industrially must pass into a more intelligent and candid recognition of grades of fitness, which should result in capitalizing hitherto unrecognized economic and social values in substandard labor. Also the value of capacity rating of the worker in one industry need not be lost in the process of transfer to another industry. Business management

should be freed from the constraint to rely upon indirect, disguised methods of executing judgments concerning personal qualifications, with a substantial saving in the costs of personnel management; and business and society might have a much more intelligent idea than they now have as to how many of the unemployed are unemployable, or employable only under specialized employment conditions.

Such a revaluation might well modify our estimates of workmen's compensation measures as instruments of economic justice. A surprisingly high proportion of all so-called industrial injuries, sometimes as high as 80 or 90 per cent., is quoted as attributable primarily to individual characteristics of workers predisposing to accidents, quite apart from any factor of circumstance over which employers can reasonably be expected to exercise any direct influence. Such a view immediately raises the possibility of some sounder apportionment of the burden of economic responsibility for injuries incurred in connection with occupation, possibly in the direction of the insurance of the constitutional hazard of the employee as well as of the administrative liability of the employer. Such a readjustment of ideas and practices might be expected to relieve much of the existing tension attending the execution of workmen's compensation laws emphasizing employers' liability.

Traditional ideas of health and disease continue to lead into frequent dilemmas arising from the inevitable interrelation of personal health with other factors of social concern. The mystery still surrounding the cause of much disease and disability; the charity still muddling our ideas of the practical care of physical misfortune; the traditional "private" nature of the dealings of physicians and patients; all these pitfalls still beset our path toward a more consistent and rational view of public health. Candid study of the physiologic and psychologic facts of employment selection, a public-private contact inevitable under our industrial system, may yield an exceedingly valuable contribution toward a better understanding of individual health and disability and other personal characteristics as matters of social concern. Such information may shed additional light upon our studies of the costs of medical care, one ironical suggestion from which thus far seems to be the economic paradox: that those who need medical care most can afford it least; and those who would buy it must have it in order to purchase it.

On the part of the individual, a keener appreciation that physical health and mental balance do, in fact, provide the surest title to a satisfying job must give finer zest to initiative to maintain health as an economic asset. For the faculties that condition human fitness in producing and enjoying the resources of our civilization—whether those qualities determine the physical and mental fitness of the manager to organize and lead, of the worker to follow leadership, of the capitalist as the wise and just trustee of civilization's treasure, or the ability of all of us to share equitably—are faculties whose essential significance and interdependence, when not distorted by factional prejudice, appeal to even the simplest economic intelligence. The opportunity for every man, woman, and child to

understand and to achieve, according to his talents, the exercise and the rewards of these faculties is a part of the unfinished work of our industrial civilization.

How confidently may we hope for a revaluation of the economic capacity and worth of individuals which can promise individual economic independence? Evidently that question is a problem of our capacity for taking intelligent social and political account of facts arising from the essential competitive nature of human relations, and a problem of our constructive capacity to modify our systems in accord with the sober convictions derived from such accounting.

The records of our United States census of manufactures from 1850 to 1927 (the year of the latest published figures) show some striking facts. During the years 1850 to 1920 the population increased some fourfold. During this seventy-year period the increase in invested capital was nearly eightyfold, the increase during the latter part of the period being considerably more rapid than that of the earlier part. The number of wage earners employed in manufacturing industries showed a ninefold increase, reaching its peak about 1920. During the same period the value of manufactured products increased about sixtyfold.

From 1920 to 1927 the population increase was about 13 per cent. After the year 1920 publication of the estimates of invested capital was discontinued; from 1900 to 1927, however, the increase in estimated horse power was about 33 per cent., certainly at least a suggestive comment upon the common complaint that machinery is displacing men. Contrasted with this 13 per cent. increase in population, with a 33 per cent. increase in mechanical horse power, and with an inestimable increase in total invested capital, following 1920 there was a marked falling off in the number of employed wage earners, amounting in 1927 to a 4 per cent. decrease from the 1920 figures. The graphic tabulations in Figure 1 make these relations more striking. The figures for 1929 and 1930 should be a picturesque addition to our previous census data.

Among the masses of census figures are others equally stimulating. To interpret these figures is to understand the industrial history of a people and to divine the future. This much seems fairly clear: Throughout the more active part of our industrial expansion the increase in the number of wage earners engaged in manufactures has never been comparable with the increase in invested capital—which is, perhaps, as accurate an index as we have of the scope of industry; while within recent years, which can hardly be accounted poor in the increase in capital investment, there has been an actual fall in the number of wage earners employed and a slight increase in the value of manufactured goods.

In other times and under other conditions of social organization, presumably unemployment always existed as the undefined misfortune of the economically less capable members of society. Our modern industrial system has defined and named this misfortune *unemployment*. Only the question of more or less seems to be dependent upon time and place and the condition of business. Industry is constantly learning new economies in the human

element of operation which have meant and seem to mean inevitably either more goods from the same number employed or the same amount of goods from fewer employed. Under the present preoccupation of industry the industrial world threatens to become an aristocracy of employment; but democracy is never unemployed; if it is not allowed its share in creating, it may take its share in destroying. America's labor leader speaks ominously of the danger in unemployed men who wish to work; Mr. Owen Young, discussing the several "surpluses" of industrial production says that "idleness of men who wish to work is the most dangerous surplus which can exist in any country"; while a recent issue of the London *Daily Mail* warns of the possible "fall of the government" upon issues growing out of unemployment.

In unemployment we seem to be confronted with the alternative of either a high tension civilization of occupational standards making use of only the best, while it casts aside substandard fitness; or a more humane civilization of employment relations adjusted to allow to every sincere desire the chance to be occupied with some

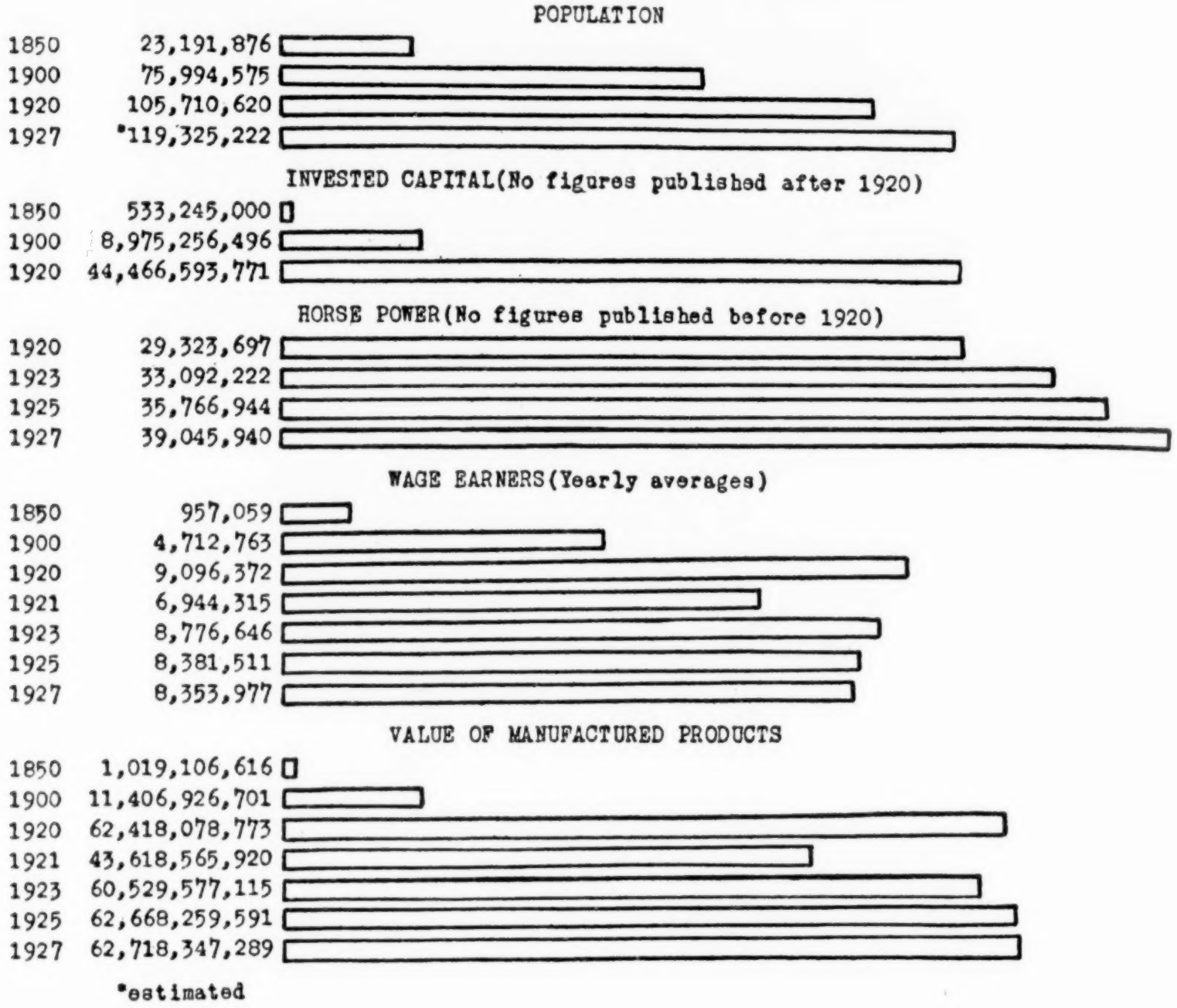

FIG. 1.—Direct tabulations from figures of the United States census of manufactures. The scales of the several diagrams are in no respect comparable with one another, having been chosen with regard only for space and for convenience in recording and reading.

however humble detail of that civilization's characteristic activities. Perhaps the conditions that determine such issues may set them beyond the deliberate choice of any individual or group of individuals. Nevertheless, it would seem that, as Americans, sooner or later we must face the facts and take some decided stand upon the question whether an aristocracy, even though it be an "aristocracy of those who work,"[2] shall dominate the most precious benefit conceived under our political democracy. For, seemingly, the outcome of such an issue must determine whether our hopeful pursuit of the "road to plenty"[3] shall prove for all of us the straight highway to a sure goal of economic independence; or for some of us only the alluring path of a splendid mirage of economic fiction.

The census records of our experience up to the present time do not go far to support confidence in *more* industry as the unique remedy for unemployment; unless it be new industry organized and operated upon lines somewhat different from those of modern competitive industry. Unfortunately for the more obvious measures to relieve unemployment, labor is not homogeneous. To continue to regard it thus in our social and governmental endeavors is to reject the lessons of years of industrial experience. Measured by the only standards with which we have practical experience, labor does not fall into two groups similar in quality and differing only in the circumstance of being employed or unemployed. Sorted by the practical experiences of industry, laborers fall into two classes: those acceptable to industry and those for the time being rejected. The dividing line between these two groups may fluctuate with circumstances; but it is always there. In this respect it seems not quite appropriate to regard unemployed labor as a surplus, considering the fact that its existence is determined by reason of having been weighed in the balance and found wanting. May not unemployed labor be regarded more properly as a by-product of industry as it exists—a by-product of unestimated social value or social menace, according as we manage it wisely or unwisely?

If our public and industrial officials are to be held responsible for administering public and private funds for subsidizing unemployment; if industrial leaders, in order to prevent or relieve unemployment, are to be expected to interfere with the natural industrial processes of selective replacement; if public works financed by public funds are to be performed by workers laid aside by private industry; or if unemployment insurance is to distribute this burden across the face of our society; as a Nation we ought to know exactly what we are trying to prevent or relieve, and to know whether the proposed means are appropriate to mere sentimental pacification, to temporary alleviation, or to thoroughgoing remedy of the fundamental difficulty. Modern public health methods accept as the foundation of all effective measures complete and reliable recording of all vital facts that provide an index to the public health. Is it extravagant to regard employment and discharge

[2] Coolidge, C.: The High Place of Labor. Foundations of the Republic, p. 76. New York, Charles Scribner's Sons, 1926.

[3] Foster, W. T., and Catchings, W.: The Road to Plenty. Boston, Houghton Mifflin Co., 1928.

as facts quite as significant to our economic and social health as are births and deaths and contagious diseases to our physical public health? The official registration of all cases of employment discharge with *actual reasons* would involve obvious difficulties. Nevertheless some provision under which industrial management might submit to a properly constituted governmental function the undisguised facts of its experience in maintaining employment standards would furnish employment information as fundamental as physical sickness and death rates in sociologic studies. The facts thus revealed should go far toward laying a foundation for practical measures sounder than the impressions and sentiments upon which sociologists and economists and government officials have hitherto been obliged to base their efforts toward the relief of unemployment.

STUDIES ON EXPERIMENTAL PNEUMONOKONIOSIS. VI. INHALATION OF ASBESTOS DUST: ITS EFFECT UPON PRIMARY TUBERCULOUS INFECTION*

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THE experimental investigation of the reaction to inhaled asbestos dust had long been contemplated as a part of the study of pneumonokoniosis carried on at the Saranac Laboratory during the past twelve years. During that time little attention had been paid to the inhalation of asbestos dust as the cause of industrial disease. It was hoped that this substance might be investigated, because it is a relatively soft and soluble silicate, the study of whose action might shed some light upon several of the vexing problems arising in connection with the inhalation of hard silicious dusts. It admirably suited our plans, therefore, to comply with a suggestion of Dr. Frederick L. Hoffman, that we undertake an experimental investigation of the reaction to this substance. At the request of Dr. Hoffman, the Asbestos Corporation of America has furnished the Laboratory with 600 pounds of exceedingly fine asbestos dust from its plant at Thetford, Quebec. The experiment was started in January, 1928, and animals have now been exposed for two years and five months. The study is not yet completed, but since so much interest has recently been manifested in the subject of asbestosis, a preliminary report is submitted at this time.

* Received for publication July 18, 1930.

ASBESTOS DUST

The dust employed in this study when dry is a light, fluffy, gray-white, stringy substance, composed of short fibers and irregular particles. Commercially this grade of asbestos is known as "King's floats." For a proper understanding of its physical characteristics, a brief consideration of its occurrence in nature and its method of preparation is necessary. The Thetford asbestos is known as chrysotile, a member of the serpentine group of minerals. Pure chrysotile is a silky, white, fibrous substance, but the presence of iron in varying states of oxidation usually gives it a greenish or greenish-brown color. It is composed of tightly packed bundles of smooth, flexible fibers, which are about 0.05 micron in diameter, and which vary from 1/8 inch to 6 inches in length. In nature these fibers occur in veins, which are embedded in hard serpentine rock. The thickness of the vein determines the length of the fibers, which run across it.

The King's floats grade of asbestos contains only short fibers which occur in very narrow veins. The hard serpentine matrix, rich in small veins, is crushed, dried, recrushed, screened and pulverized, and finally passed over shaking screens from which the fibers are withdrawn by suction. The material is then graded by screening, and the fraction which passes the finest screen is allowed to settle in a large room. Such material (King's floats) contains not only short fibers, but in addition many small particles of serpentine rock.

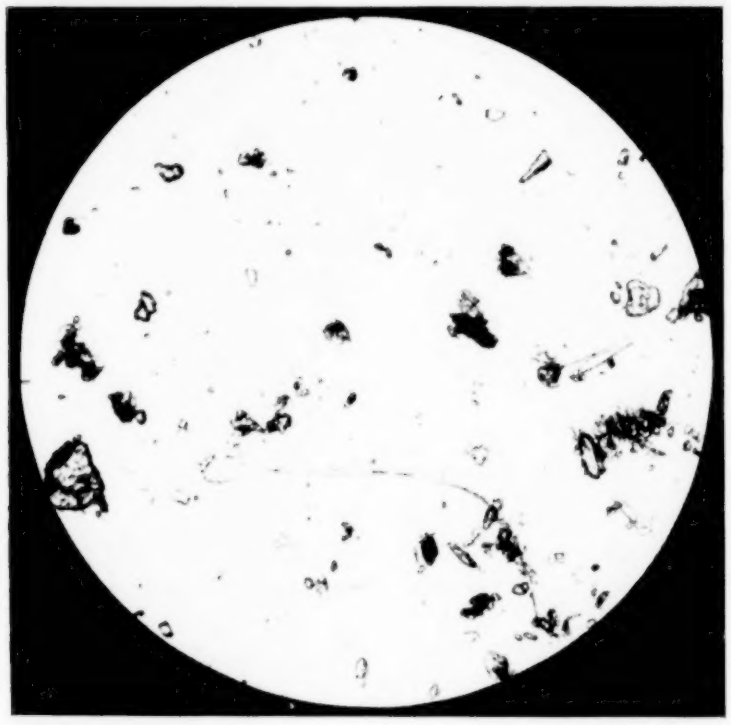

FIG. 1.—Asbestos dust. ×90.

Microscopic examination reveals the fact that particulate matter is far in excess of fibrous material. The particles vary in size from 10 microns to 0.5 micron in diameter. The fibrous traction is less uniform. In length the fibers vary from 1 mm. to 1 micron or less. Many isolated, long, smooth, thin, flexible strands are seen, while others consist of short thick bundles with broken ends. The fibers are highly refractile when viewed by polarized light. (See Fig 1.)

The dust has a specific gravity of 2.26. It loses only about 1 per cent. of its weight when heated at 110°C., yet in a moist atmosphere sufficient water is absorbed to increase noticeably its tendency to clumping or matting.

In chemical composition, pure chrysotile is chiefly composed of crystalline hydrated silicate of magnesium ($3MgO \cdot 2SiO_2 \cdot 2H_2O$). It is, however, seldom free from impurities which partially replace the magnesium to form more complex silicates. Ferrous iron is the most common contaminant, and it may sometimes constitute as much as 10 per cent. of the mineral as it occurs in nature. A portion of this iron may be oxidized to the ferric state.

The following analysis has been made from samples of the dust used in the study:

	%
Total SiO_2	39.32[1]
Fe_2O_3, Al_2O_3	8.84
CaO	0.67
MgO	35.56
Alkalies and loss	2.87
Combined water	12.74

[1] Free SiO_2 (by rational analysis) = 2.6%.

The material reported as aluminium oxide and ferric oxide was largely the latter. Preparations of the dust stained with potassium ferricyanide and dilute hydrochloric acid indicate that there is ferrous iron in the majority of the fibers; a few exhibit irregular staining with ferrocyanide.

Anderson and Clark (1) have shown that treatment of Thetford chrysotile with hydrochloric acid completely destroys the typical crystalline pattern

of the material produced in Bragg's X-ray diffraction technic.

Careful and repeated search has failed to disclose any yellow structures resembling asbestosis bodies in the dust previous to its residence in the animal. The appearance and method of development of these structures will be discussed later.

Experimental Procedures

The general plan of the experiment is the same as that previously employed in studies on granite (2), marble (3), carborundum (4), and quartz (5). In the present instance, animals are kept in cages along the walls of a room 6 by 8 feet, and 8 feet high, provided with two 3 by 5 foot windows. A cloud of dust is maintained in the atmosphere of this room by apparatus located in an alcove, 6 feet from the nearest animal. This apparatus consists in a horizontal drum 2 1/2 feet long and 1 1/2 feet in diameter, placed 8 inches above the floor. Its top is open, and in it rotates a metal paddle, whose shaft projects through the end of the drum and then through the partition into an adjacent room in which is located an electric motor. By properly selected pulleys and countershafting, the speed is so regulated that the desired dust concentration can be maintained. Ventilation is secured by opening one of the windows an inch or more from the top. The animals are kept, generally in pairs, in 12 by 12 by 12 inch wire cages set on racks at the sides and back of the room. While it will be shown that the dust concentration progressively decreases from the floor to the ceiling, this factor is compensated by frequently changing the position of the cages on the racks.

As measured by the operation of the motor, the daily period of exposure is eight hours, but since the animals remain in this room during the night, the actual exposure is much longer, considerable time being required for all the dust in the atmosphere to settle. In warm weather the period is somewhat shortened by opening the windows when the motor is stopped in the evening. From the commencement of the experiment on Jan. 20, 1928, until the present date, the dust exposures have been continued for eight hours, on six days a week, with the exception of a period of three weeks in the month of January, 1930, when the motor was being repaired.

Dust Concentration

The amount of dust in the atmosphere of this room has been checked with a Greenburg-Smith impinger apparatus.[1] Samples taken on Feb. 14, 1928, one month after the experiment was started, showed the concentrations given in Table 1.

The rate of suction on the sampling apparatus was 1 cubic foot per minute; the sampling times were fifteen minutes and thirty minutes. Owing to the fibrous nature of the dust, the particles were not filtered before the counts were made, but the very large particles (those greater than 100 microns) were simply neglected in counting. Particles over 1.5 microns in diameter, magnified ninety-five times, were counted in the usual manner in a Sedgewick-Rafter cell,

[1] Appreciation is expressed to Mr. Morris Dworski for making these dust determinations.

after being allowed to settle for thirty minutes. The smaller ones varying from 1.5 microns to 0.5 micron in diameter were counted in a hemocytometer chamber at a magnification of 410. The usual blank controls on the distilled water used to collect the dust samples were made, and the number of accidental particles was subtracted before the results were tabulated.

This concentration was maintained with little variation until January, 1930, when the speed of the paddle was accelerated, and the amount of suspended dust was increased approximately ten times. The majority of the animals to be discussed in this report, however, were subjected only to the smaller dust concentration.

In this experiment 128 guinea-pigs, eighteen white rats, and seven rabbits have thus far been used. A portion of the guinea-pigs were infected with tubercle bacilli in an attempt to determine the influence of this dust upon a tuberculous process.

One group of sixty-three guinea-pigs were infected with tubercle bacilli four days before the dust inhalation was begun; forty of them were placed in the dusting room and twenty-three were kept in a normal atmosphere as controls. Two years after the experiment was started, twelve dusted and twelve normal guinea-pigs were infected with tubercle bacilli. This group was then set aside without further exposure to the dust. All the rats and rabbits and fifty-four of the guinea-pigs were used in studying the uncomplicated effects of inhaled asbestos dust. Each of the various groups will be described separately.

TABLE 1.—DUST COUNTS

COUNT NO.	HEIGHT OF INTAKE FROM FLOOR	MILLION PARTICLES PER CUBIC FOOT OF AIR				
		A 100–10μ (× 95)	B 10–1.5μ (× 95)	C 10–0.5μ (× 410)	D 1.5μ or less (C − B)	Total (A + C)
	ft.					
1	2	1.130	8.033	56.666	48.633	57.796
2	2	1.258	6.625	51.600	44.975	52.858
Average		1.194	7.329	54.133	46.804	55.645
3	4	0.450	4.960	35.000	30.040	35.450
4	4	0.400	4.440	43.333	38.893	43.733
Average		0.425	4.700	39.166	34.466	39.591

Pathologic Lesions

Non-Tuberculous Guinea-Pigs

Up to the present date, twenty-nine months after the commencement of the dust inhalation, fourteen animals, or 26 per cent. of the group, have died of epizootic pneumonia, and fifteen more succumbed to other conditions, such as enteritis and peritonitis. As this experiment was designed to demonstrate the maximum effects which could be produced by the inhaled dust, relatively few of the ani-

...als have been killed. A number ...ave, however, been sacrificed at 30, ...0, 90, 354, 610, 782, and 847 days. ...ommencing with the eighth month ...f exposure, the effects of the dust ...ave also been followed radiographically in the living animals.

The pulmonary localization of inhaled asbestos differs from that of any other dust thus far studied. Granite, quartz, carborundum, and soft coal particles penetrate the terminal air spaces of the lung, and large quantities ultimately localize in the subpleural alveoli. Asbestos dust, presumably because it is so largely composed of elongated fibers, is carried by the inspired air only to the distal respiratory bronchioles, and there the major portion of the material comes to rest. Mononuclear phagocytes from the adjacent connective tissues enter the lumen of the air passages and engulf the particles. The phagocytes with their contained fragments of dust are frequently pushed aside into the alveoli which pouch out of the sides of the bronchioles, where many of them remain indefinitely. More dust entering the lung is held up by the partial obstruction created, and further reaction is largely proximal to the point of original localization. A few particles pass beyond into the proximal portions of the alveolar ducts, but in the majority of cases the deposition of asbestos dust occurs within respiratory bronchioles of the second and third orders and in the alveoli given off directly from those structures. (Fig. 2.)

In guinea-pigs killed thirty days after starting the dust inhalation, occasional small mononuclear phagocytes containing a number of brown or black particles and a few short spicules of doubly refractile material are visible in the alveoli budding off from the respiratory bronchioles. A few multinucleated dust cells are encountered. The lymphoid tissues of the bronchial tree exhibit a moderate degree of hyperplasia. The lymphatic trunks coursing through the bronchial walls are wide open, but no dust cells or other material can be found in

FIG. 2.—Cross section of lungs of guinea-pig exposed to asbestos dust inhalation 840 days. Normal size.

FIG. 3.—Roentgenogram of guinea-pig exposed 880 days to asbestos inhalation.

their lumina. The tracheobronchial lymph nodes exhibit no more dust containing cells than those in any normal animal kept in a laboratory atmosphere.

At sixty days, the number and size of the dust cells in the terminal respiratory bronchioles is considerably increased. The lumina of some of the lateral alveoli are completely blocked by masses of mononuclear phagocytes and larger giant cells. Some of the latter have six or more nuclei. Frequently in Zenker-fixed tissues, these phagocytes have a diffuse yellow-brown color which is presumably due to the presence of dissolved iron in their cytoplasm. In tissues fixed in formalin and stained with acidified potassium ferrocyanide, such cells exhibit a diffuse blue coloration. In addition, there are certain particles in their cytoplasm stained very deep blue which are probably the source from which the iron is dissolved. Doubly refractile intracellular spicules are numerous; they are most readily demonstrated in polarized light. After sixty days' exposure to dust, the first evidence of that peculiar structure which has been called a "curious body" is detected. We prefer to adopt Stewart and Haddow's (6) designation of "asbestosis body," and shall use the latter term exclusively.

After two months the number of asbestosis bodies is small and they are atypical in appearance. The most common form is a club-shaped swelling at the end of a smooth pale greenish fiber. Some present small fusiform swellings along the course of a short fiber. They are seen almost without exception inside of large giant cells.

After ninety days' exposure, the number of these structures has very markedly increased, and they have assumed their typical form. The smaller ones are still contained in giant cells, but many long forms appear to be free in the stroma of the lung. The amount of cellular reaction has not kept pace with the number and size of the foreign bodies. It still consists of collections of mononuclear and giant phagocytes within the lumen of a localized group of air spaces, together with a very slight infiltration of the adjacent septa. Hyperplasia has occurred in the lymphoid tissues associated with the bronchi proximal to the foci of maximum dust reaction, but no phagocytes have carried dust particles into the lymph nodules. The lymphatic trunks are widely distended, but they contain no cells or dust particles. The tracheobronchial lymph nodes also show well-marked hyperplasia, but the number of dust containing cells is still small.

During the next four months, no animals were killed, but nine died of various accidental causes. In them it is found that increasing quantities of dust are accumulating in the vicinity of the respiratory bronchioles, and that the amount of cellular reaction is somewhat increased. Asbestosis bodies are so extremely abundant that as many as eight have been encountered in an oil immersion field. They are practically absent in other portions of the lung. The other changes in the lymphoid tissues and lymphatic vessels remain much the same. There is as yet no evidence that appreciable amounts of dust are being transported to the tracheobronchial lymph nodes.

In three guinea-pigs killed on the 357th day, the amount of reaction has

veloped to such a degree as to be isible on gross section of the fixed

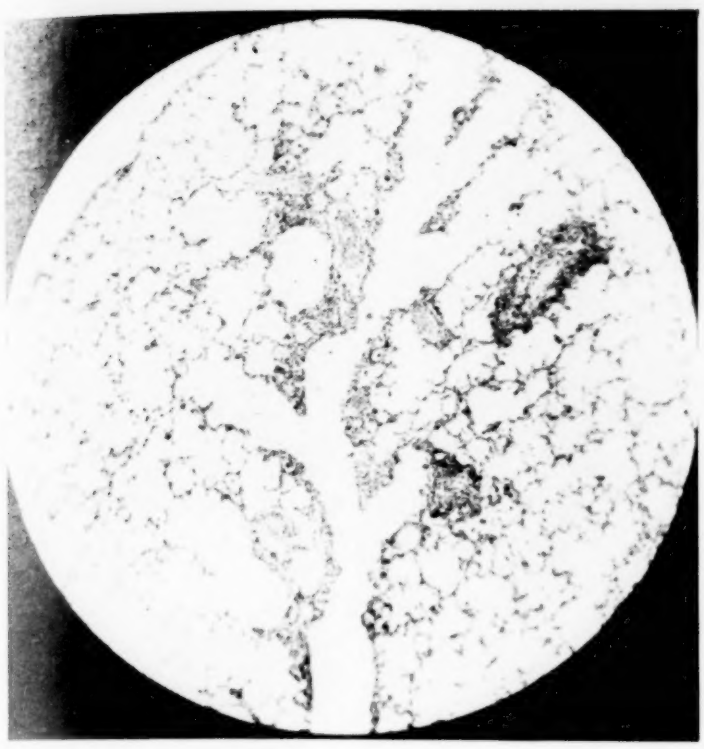

FIG. 4.—Longitudinal section of bronchus and its peripheral branches. Exposure 357 days. Approximately ×45. Note that dust reaction is localized to region of respiratory bronchioles. No reaction in lymphoid tissues.

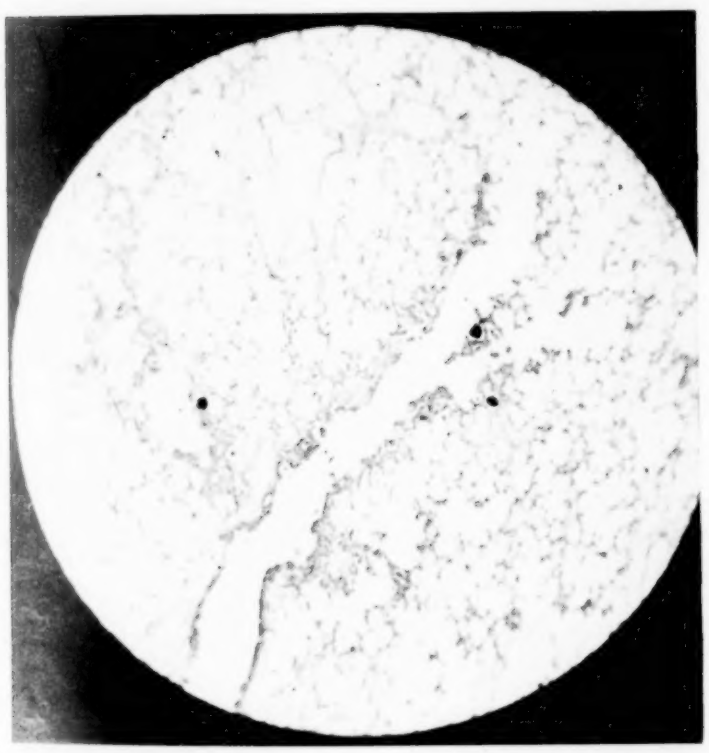

FIG. 5.—Longitudinal section of bronchus and its peripheral branches. Exposure 436 days. Approximately ×45. Note that dust reaction is localized to region of respiratory bronchioles.

tissue. Uniformly distributed over the entire surface of both lungs are fine yellowish-white ovoid flecks of granular appearance with ill-defined borders. They are arranged radially on the surface and suggest a relationship to the

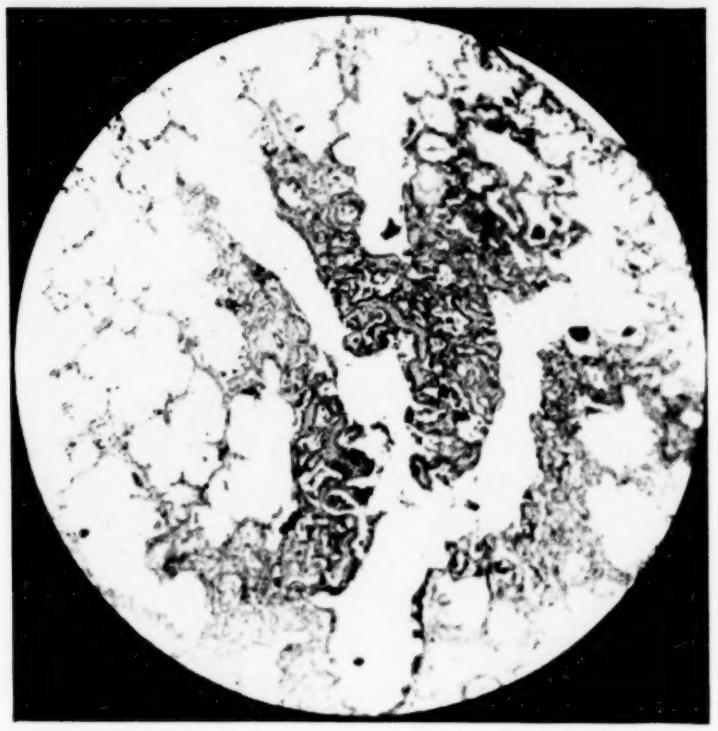

FIG. 6.—Peribronchiolar dust reaction in normal guinea-pig exposed 840 days. Connective tissue stain. Approximately ×90.

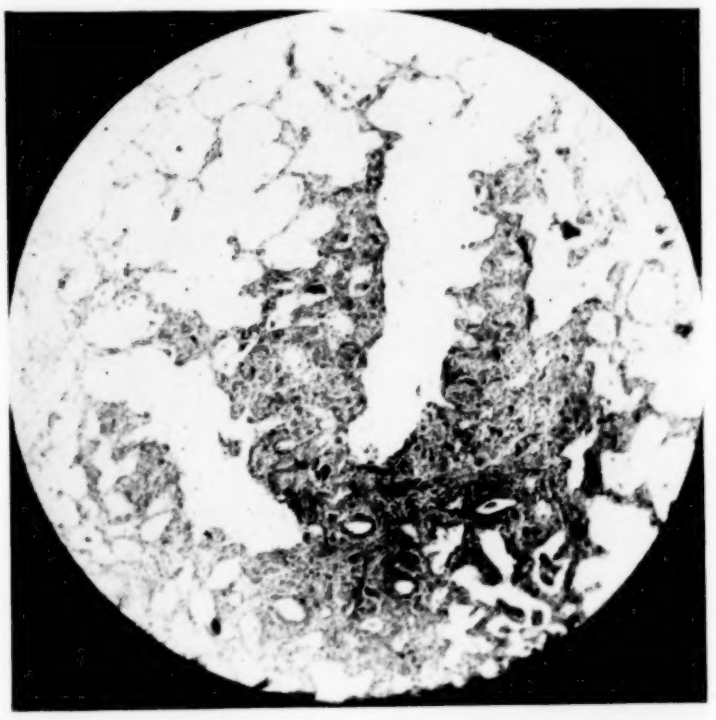

FIG. 7.—Early tuberculous reaction in focus of asbestosis; endogenous extension 357 days after infection. Approximately ×90.

finer bronchioles. All are of approximately the same size and none exceed 1 mm. in diameter. Microscopically those foci are similar in every way to

the type of reaction already described. The walls of the bronchioles, proximal to alveolar ducts, are thick, but this is due solely to an excessive local accumulation of phagocytes. There is no evidence that fibroblasts are proliferating. The dust cells have not migrated to any extent from the original foci of localization; none are found in the pulmonary lymphoid tissues, but some have been transported to the tracheobronchial lymph nodes. (Figs. 4–7.) In the medullary sinuses of the latter location there are now definite clusters of brown-stained iron containing phagocytes. In their cytoplasm many irregular black or brown particles and refractile elongated spicules are recognizable. Occasionally a giant cell is seen partially surrounding a very long fiber which may project beyond the limits of the cell. One such fiber measured 25 microns in length. No asbestosis bodies are seen. As yet the only evidence of reaction in these nodes consists in an active proliferation of the follicle cells.

While asbestosis bodies cannot yet be found in the lymphoid tissues, they do occur outside the lung. Two of the 357-day animals had developed a chronic fibrous pleurisy, and occasionally in the connective tissue there are small clusters of phagocytes containing dust and asbestosis bodies. In an isolated field, five of them have been counted.

On the 449th day, two asbestosis bodies are found within a giant cell in the tracheobronchial lymph nodes.

All animals killed or dying on and after the 505th day are characterized by the *proliferation of fibroblasts*. This reaction is confined to the walls of the air spaces adjacent to the deposits of accumulated dust phagocytes. At first only a few cells are involved, but after still another 100 days, a true fibrosis, accompanied by the formation of very considerable amounts of collagen, has developed. The walls become progressively thicker, and encroach upon the lumen of the air spaces. The epithelium of the latter contracts and becomes cuboidal, so that the area of reaction resembles a gland with a heavy connective tissue stroma. The lumina of these gland-like atelectatic air spaces are more or less completely filled with compact masses of dust cells and brown-stained giant phagocytes whose cytoplasm is gorged with dust particles, fibers, and asbestosis bodies. In the thickened septums there are many of the latter, which are generally extracellular. (Figs. 6, 9, and 10.)

The amount of dust carried into the tracheobronchial lymph nodes now becomes more abundant. It consists largely of amorphous or granular black or brown particles with many doubly refractile spicules which fail to stain specifically for iron. Occasionally a few giant cells, sometimes in small clusters, contain typical asbestosis bodies. The stroma of the nodes about the masses of dust cells now exhibits a limited amount of fibroblastic proliferation.

The latest stages of the reaction of asbestos dust thus far produced are manifested in the lungs of five guinea-pigs, of which three were killed on the 782nd day, and one on the 840th day. All five exhibited a large number of subpleural gray-white nodules at least 1 mm. in diameter, uniformly distributed over the surface of the lung.

section of the organs, sharply demarcated ovoid areas are disclosed in every part of the lung. One animal shows a fibrous pleurisy over

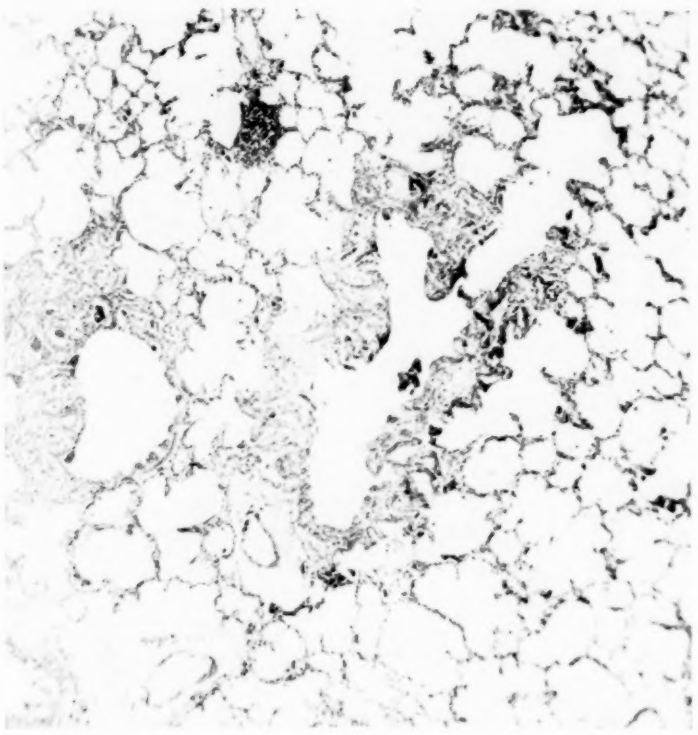

FIG. 8.—Peribronchiolar focus of asbestosis; guinea-pig exposed 782 days. Approximately ×70.

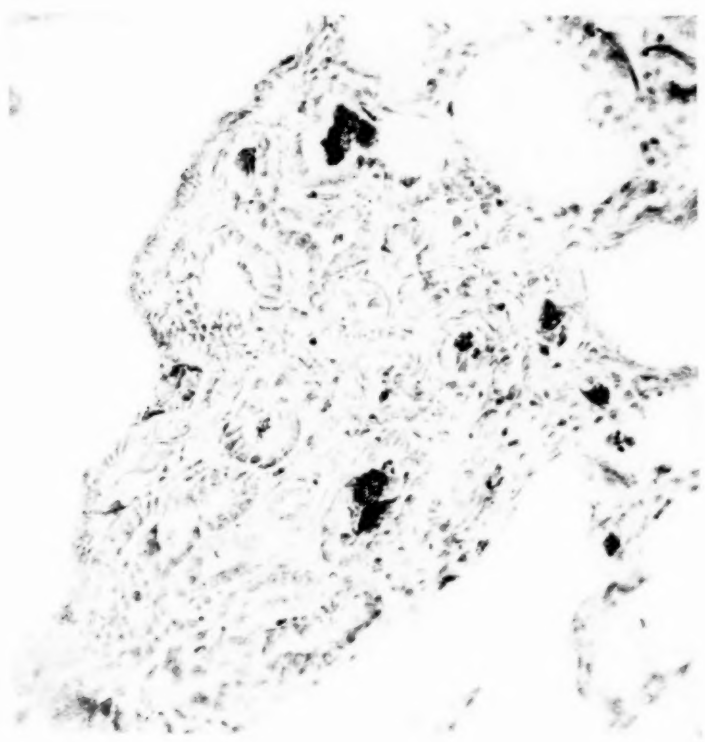

FIG. 9.—Magnified field of Figure 8. Note gland-like air spaces. Approximately ×140.

portions of the lung without evidence of an underlying pneumonia. The tracheobronchial lymph nodes are deeply pigmented in their central portions.

The first radiographic examination to demonstrate definite changes in the lung was obtained after a dust expo-

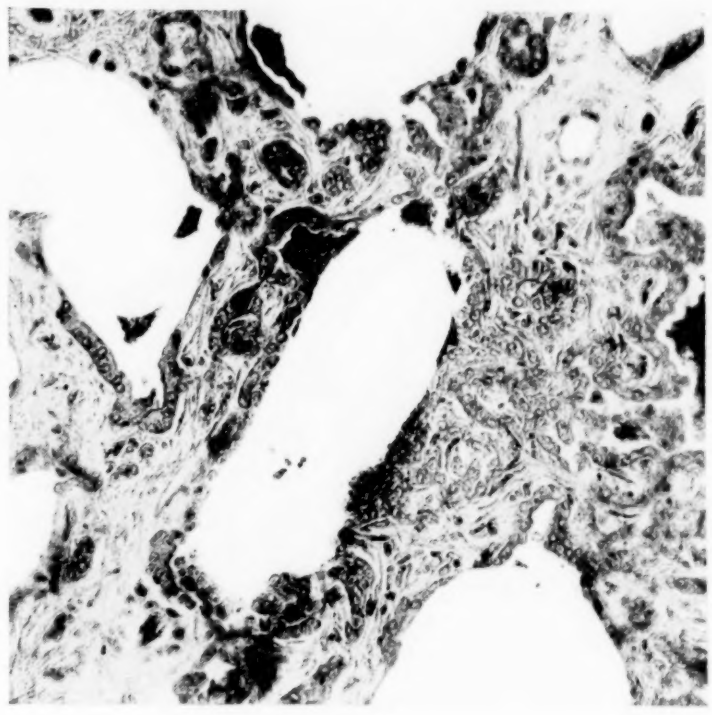

FIG. 10.—Magnified field of Figure 8. Connective tissue stain. Note strands of collagen between gland-like alveoli. Approximately ×140.

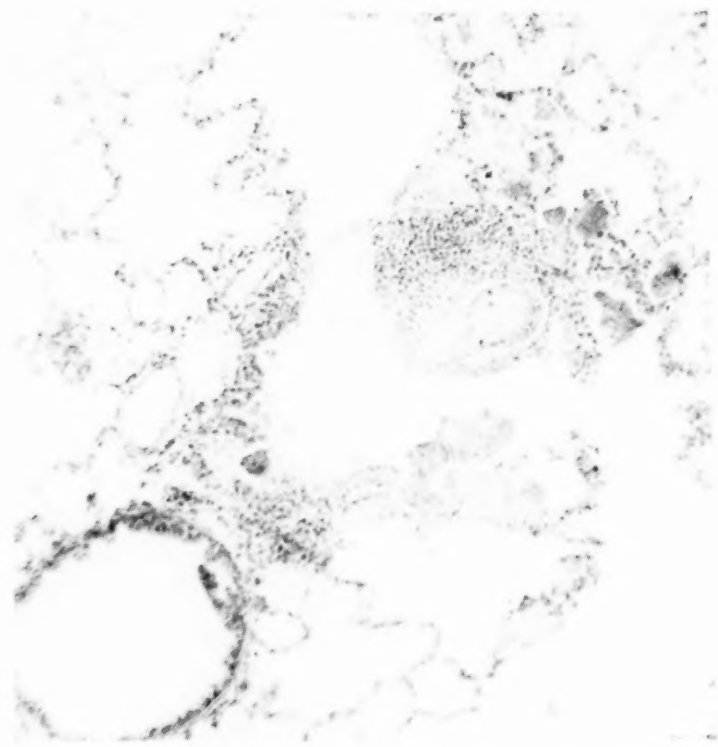

FIG. 11.—Asbestosis in rabbit exposed 149 days. Approximately ×140. Note giant cells, one of which has invaded nodule of lymphoid tissue.

sure of two years. At this time the presence of very fine mottling was detected in the lower portions of the lung fields. After another five months

had elapsed the changes had become much more marked. As illustrated in Figure 3, they consist of strands of coarse mottling which radiate outward and downward from the hilum. The outlines of the mediastinum and the pericardium are obscured by similar shadows. Whether the apexes are also involved cannot be definitely determined.

Pathologic changes in the lungs of experimental animals are much more readily demonstrated radiographically if the lungs are removed from the thorax and inflated before exposure to the X-ray. Roentgenograms have been made in this manner of animals with well-developed asbestosis and the shadows produced are characterized by a pronounced thickening of the linear markings which extend outward from along the main bronchi to the margins of the lung.

Microscopically, the lungs present lesions similar to those already described, but they are more extensive and fibrosis has become more marked. In stains for connective tissue, abundant collagen formation is now apparent in the walls of the respiratory bronchioles and their alveoli. Dust cells and asbestosis bodies are very abundant. The extension of the process from the terminal bronchioles proximally into respiratory bronchioles of greater diameter is obvious.

In addition to these characteristic changes, the lung of one animal exhibits more or less circumscribed foci of fibrosis in the alveolar septums peripheral to the usual reaction zone. The lumina of the included air spaces are normal in size, and apparently fully distended. Their walls are lined with easily visible cuboidal or flattened epithelium which occasionally dips into the thickened septums to form gland-like structures. In marked contrast to the previously mentioned areas of fibrosis in the respiratory bronchioles, these more peripheral foci include practically no dust cells or asbestosis bodies. One lobe of this lung is covered by organized fibrous pleurisy which also fails to exhibit dust deposits.

Examination of the trachea and stem bronchi, both by direct and by polarized light has failed to demonstrate appreciable amounts of dust or local reaction. The only evidence of irritation is the presence of an occasional polynuclear leukocyte on the ciliated surface and the distention of the goblet cells with mucus. More peripherally the connective tissue coats of the bronchi of the second and third orders exhibit considerable infiltration with lymphoid cells, and some engorgement of the blood vessels. The other viscera, the spleen, liver, kidneys, pancreas, and abdominal lymph nodes have been uniformly free of dust or evidence of reaction attributable to it.

In the guinea-pig, the development of the early (two years and five months) response to inhaled asbestos dust may be summarized as follows:

The particles and elongated fibers of asbestos dust do not, during this period, penetrate so deeply into the air passages of the lung as do the other dusts previously studied. The major portion is held up and phagocytosed in the lumina of respiratory bronchioles and the alveoli given off from their walls. Increasingly large masses of phagocytes accumulate in the lumina of these air spaces, and many of the cells migrate into the framework of

the adjacent lung tissue. In this location there develops a low grade chronic inflammatory reaction characterized by infiltration with monocytes and a few lymphoid cells. Approximately 500 days after the commencement of dust inhalation, the connective tissue cells in the walls of the bronchiole and its alveoli begin to proliferate.

Thereafter, the reaction is characterized by increasing amounts of fibrous tissue which develops in the immediate vicinity of the dust cells. The thickening stroma encroaches upon the lumen of the air spaces whose epithelium, being compressed, appears cuboidal, as is the case in pulmonary atelectasis. The picture produced somewhat resembles glandular tissue. In addition to this lesion, which is characteristic of all the animals examined, one of three of the last guinea-pigs killed showed a further change consisting in focal areas of fibrosis in the alveolar septums peripheral in the pulmonary unit to the point of dust localization.

Changes in other structures of the lung are not striking. There is well-marked hyperplasia of the lymphoid tissues in the periphery of the lung, but dust containing phagocytes do not migrate toward and enter these masses, as is the case with quartz inhalation. Dilatation of the large lymphatic vessels in the connective tissues of the bronchi and blood vessels has been a constant finding in all the animals of the series. Even thirty days of dust inhalation would appear sufficient to produce this effect. Its cause has not been discovered; thrombi within the lumen of the lymph vessels are lacking, and a central obstructive lesion in the tracheobronchial lymph node is a late development. Only after 700 days do fibroblasts begin to proliferate in the medulla of the nodes.

Asbestosis bodies have not been found in any animal killed before sixty days of dust exposure. When first discovered, they are small, somewhat atypical in form, and generally contained within the cytoplasm of phagocytes. Within the next sixty days they rapidly increase in number, size, and complexity of structure. The larger ones appear to be liberated from the cells, and ultimately to be free in the air passages or intracellular spaces. In most cases, these structures, presumably because of their size, are not transported from the point where they first develop in the lung. Occasionally they have been found in the tracheobronchial lymph nodes and in areas of chronic fibrous pleurisy. In these instances they are usually intracellular.

Rabbits

Five rabbits exposed to asbestos dust for 40, 53, 149, 253, and 330 days, respectively, have thus far been killed and studied. Within the period of time included, the reaction to the inhaled dust has been characterized by increasingly large focal collections of mononuclear and giant phagocytes. These, as in the guinea-pig, tend to accumulate in and along the respiratory bronchioles, although in the last two animals killed, some collections of dust cells have been seen in the more peripheral air spaces. In marked contrast to their behavior in the guinea-pigs, the phagocytes exhibit a tendency to migrate into masses of lymphoid tissue. This occurs early, and has been observed in all but the

first animal killed. It is probably due to anatomic differences in the two species. In the rabbit, abundant deposits of tonsil-like lymphoid tissue are situated directly beneath the epithelium throughout the course of the bronchial tree; in the guinea-pig the lymphoid nodules are not so readily accessible. The phagocytes are filled with great quantities of doubly refractile dust spicules, but *no typical asbestosis bodies have yet been discovered.* When stained for iron, none of the fibers in the first four animals reacted. In smears of lung tissue from the last animal killed on the 330th day, a few elongated fibers exhibited irregular deposits of both ferric and ferrous iron, and there were occasional globular swellings along their course. But these fibers did not present the usual golden-yellow color of an asbestosis body, and the alterations in structure were in no way characteristic.

No suggestion of fibrosis or even reaction in the framework of the lung adjacent to the collection of dust cells has been discovered, but it is still too early to expect much change. In the guinea-pig, the first indication of fibrosis was found on the 500th day.

Albino Rats

Repeated attempts to study the reaction to inhaled asbestos in the white rat have been defeated by the development of epizootic pneumonia, often complicated by abscess formation. The first series was composed of twelve animals, all of which succumbed to this infection. One survived for 450 days, but in all of them the reaction to chronic pneumonia so obscured the picture that no deductions were possible. In December, 1929, six new, apparently healthy rats were placed in the dust room. During a period of 118 days three have been sacrificed and one has died. All of them have had pulmonary abscesses. In only one animal, which was killed on the seventieth day, could asbestosis bodies be detected, and in this case prolonged search of smears of the lung tissue stained for iron has revealed only two of these structures. One was very small, but typical in appearance. The other consisted of a bundle of fibers frayed at the end, and upon one of the fibrils were a few irregular swellings. In another rat killed after 118 days, no asbestosis bodies have been found.

The Asbestosis Body

Since the development of the peculiar structure termed an asbestosis body in the lungs of animals compelled to inhale asbestos dust may have an important bearing upon the general problem of pneumonokoniosis, it deserves detailed consideration.

The forms encountered in the lungs of guinea-pigs in every way resemble those pictured in the human cases. While individual specimens exhibit marked variations, there are certain features which are characteristic of all the forms observed. The fully developed body consists of a brilliant golden-yellow, beaded or haustral rod, which may be either straight or curved. Its color resembles that of the hemosiderin deposits found in areas in which hemorrhage has occurred. In length they vary from 1 to 250 microns, and in width from 0.5 micron to 10 microns. The shorter ones are usually straight, but the larger forms are curved, or even coiled. They do not stain with any of the dyes ordinarily used for

issues, but Wright's blood stain is absorbed to some extent, giving them a greenish cast. Fixing fluids and the usual histologic stains have no apparent effect upon them. Iron in the ferric state may be demonstrated by

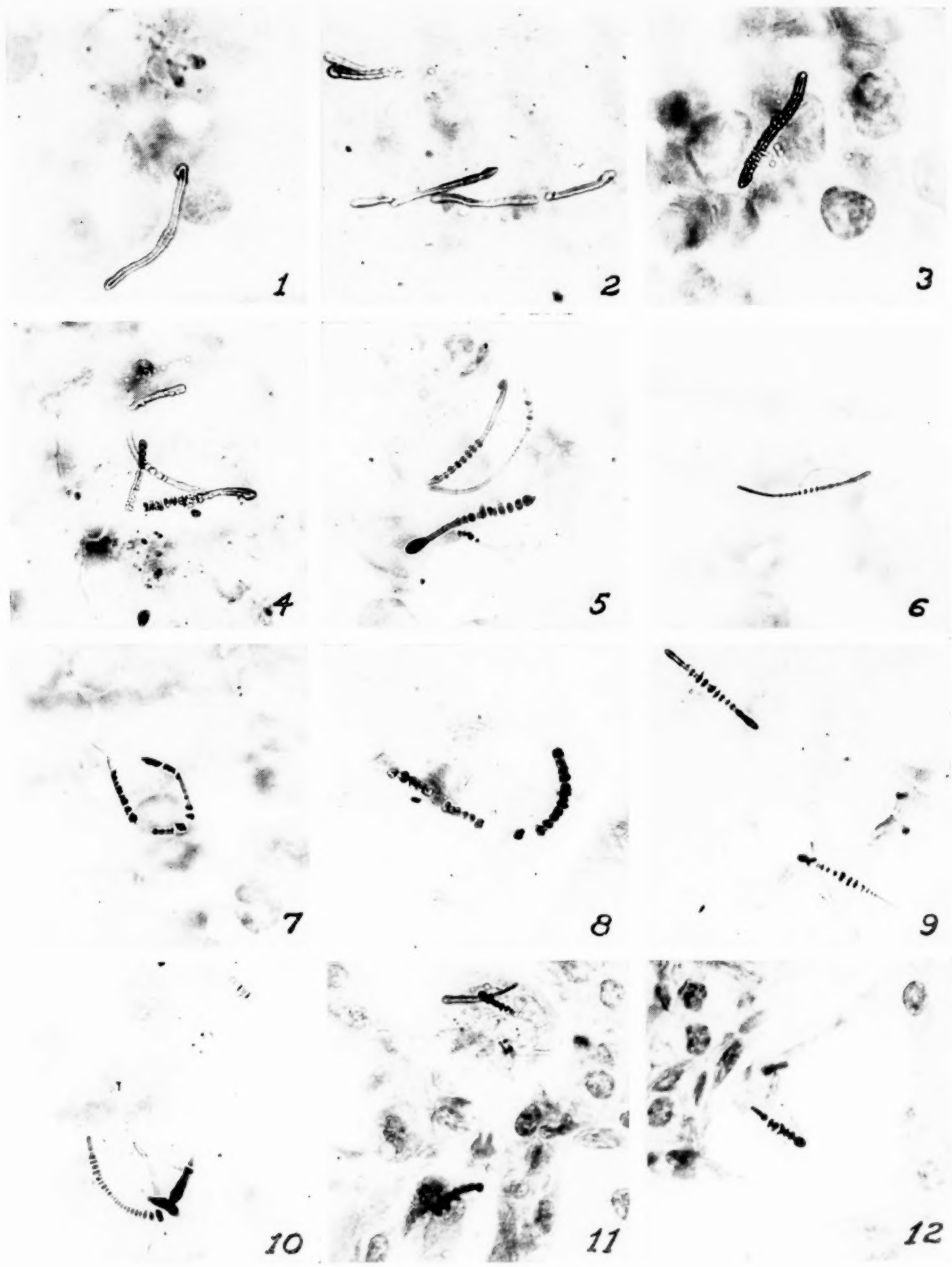

FIG. 12.—Asbestosis bodies in guinea-pigs' lungs. Nos. 1 to 10 from smears of lung tissues; Nos. 11 and 12 in sections. Approximately ×560.

No. 1 shows a smoothly coated fiber; in No. 2, no fiber is visible; No. 7 shows coating material cracked along fiber. Other illustrations show various forms of this structure.

treatment with potassium ferrocyanide in dilute hydrochloric acid (McDonald and Stewart). Examined under polarized light, they are not doubly refractile. The shorter forms are usually found within the cytoplasm of a foreign body giant cell; longer ones may be coiled within such cells, but more usually they lie free in the lumen of an alveolus or in the intercellular spaces of the pulmonary framework. Whether they actually exist in the free state during life or whether, owing to their elasticity, they are set free in the process of preparing sections and smears, cannot be determined. (Fig. 12.)

The lung of the rabbit apparently offers a much less favorable environment for the production of the asbestosis body than that of the human being or the guinea-pig. Nothing faintly resembling one of these structures has been found until the dust exposure has been continued for a period of eleven months. At that time, in hematoxylin and eosin preparations occasional fibers are seen which have a muddy, greenish

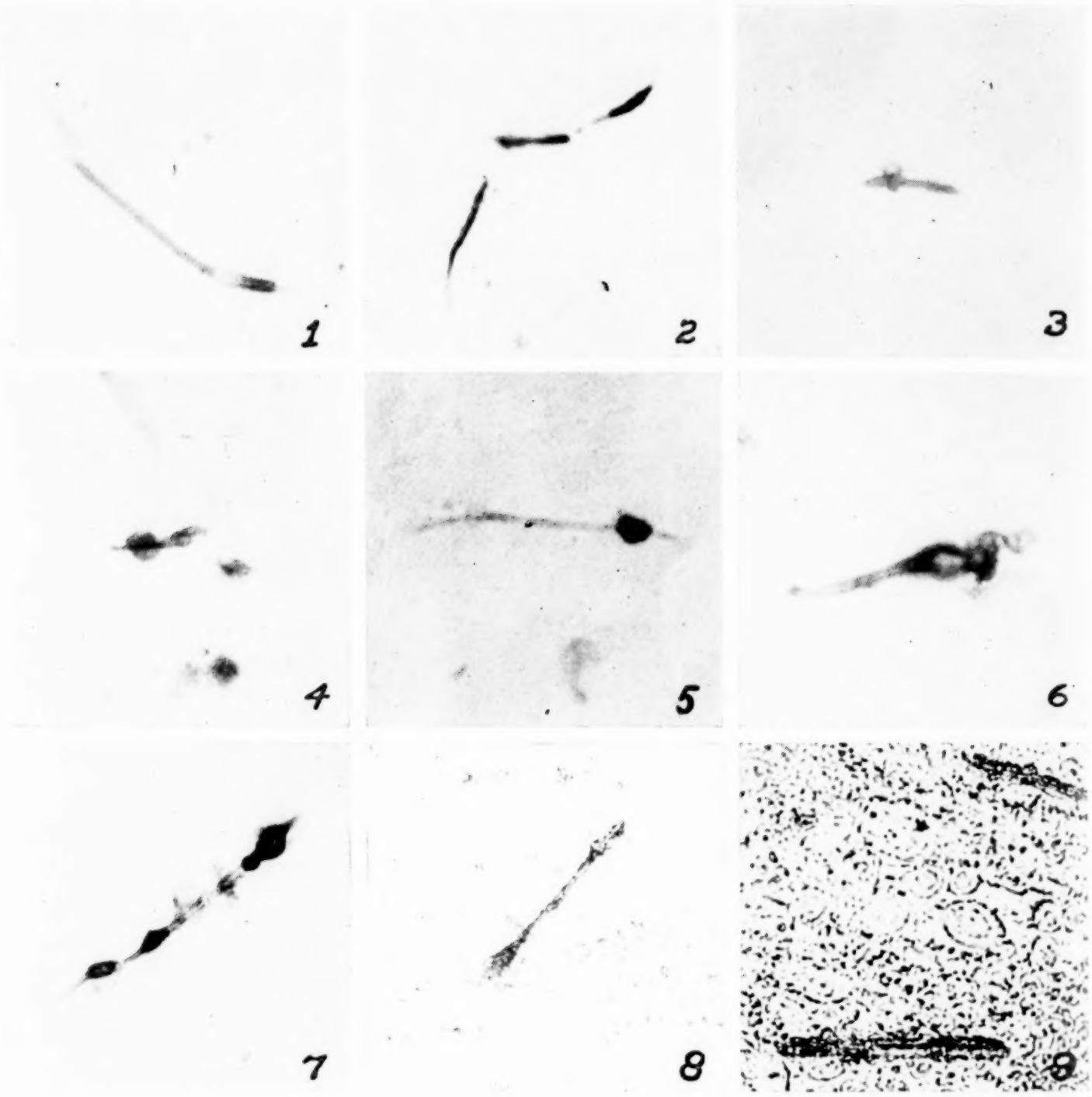

FIG. 13.—Atypical asbestosis bodies from lung of rabbit exposed 330 days. Nos. 8 and 9 are artificially produced asbestosis bodies. ×840.

lue, but they do not exhibit the regular beading, or the characteristic golden-yellow color seen in the guinea-pig. Preparations stained for either ferrous or ferric iron demonstrate a few very long fibers containing irregular swellings of a deep blue color. Whether typical asbestosis bodies will ever develop in this animal remains to be demonstrated. (Fig. 13, Nos. 3 and 4.)

In the lung of the white rat they would also appear to be very uncommon, although the widespread incidence of chronic pulmonary infection among the animals thus far exposed, in a measure invalidates the results. Thus far, only two asbestosis bodies have been discovered in one animal of this species. If the environment were favorable, it would seem probable that more of these bodies would have been detected, even in the presence of complicating pneumonia.

Our observations have led us to believe that the asbestosis body develops as the result of chemical changes taking place within or about inhaled asbestos fibers. The dust previous to contact with the pulmonary tissues shows no such structures. A period of time which varies with different species of animals must elapse after the fibers have entered the lung, before the asbestosis body makes its appearance. The first forms encountered are different from those seen later. They consist of iron containing, sheath-like or nodular swellings along the sides or at the ends of a fiber. From such points further extensions occur until in some cases the whole filament acquires a uniform coating which is brittle (Fig. 12, No. 1). Movement or other stress causes the coating substance to crack, separating it into segments (Fig. 12, No. 7).

Chemical action continues and the segments tend to elongate in a direction at right angles to that of the original fiber. Extension also takes place at the ends of the fiber, so that it increases in length. Small fragments may also serve as nuclei for chemical activity; about them spherical or ovoid masses develop. The resultant forms exhibit many variations. Perhaps the most common ones consist of an axial fiber traversed at intervals by a variable number of thickened crossbars. Often one end and sometimes both ends are swollen and spindle shaped. In other instances, the central fiber cannot be discovered (possibly it has been dissolved?); the body then consists of a row of globules. Sometimes these are of uniform diameter; frequently they decrease progressively from one end to the other. In guinea-pigs exposed for more than two years, many bizarre branching globoid forms are seen, which resemble clusters of tuberous bulbs.

An observation which merits comment is the scarcity of asbestosis bodies in the tracheobronchial lymph nodes. Presumably because of their size, the majority of the inhaled asbestos fibers are not transported to any distance by phagocytes. Nevertheless, after exposure to the dust has been continued for somewhat over a year, appreciable numbers of doubly refractile non-iron-staining fibers can be found in the lymph nodes of the mediastinum. They are embedded in the cytoplasm of phagocytes which have migrated from the lung. After two years, large masses of such dust cells have accumulated, and yet asbes-

tosis bodies are not found in any number in the tracheobronchial lymph nodes. Those which do occur are located in clumps of giant cells. It is believed that the formation of the few asbestosis bodies found took place in the lung, and that they were then transported to the lymph nodes in emboli of phagocytes.

Why do asbestosis bodies occur so rarely in the material which has been transported to the lymph nodes? Were the fibers during their period of residence in the lung deprived, by solution, of iron or some other substance necessary to their production; or are the transported fibers essentially different from those remaining in the lung; or, again, is the environment furnished in the lung especially favorable for the production of asbestosis bodies?

To answer the last possibility, injections of asbestos dust have been made into other portions of the body. In the peritoneal cavity of the guinea-pig attempts to produce asbestosis bodies have met with but slight success. In one series each guinea-pig was injected with 2 c.c. of a heavy suspension of the King's floats dust, which was so prepared by sedimentation (7) that only particles varying from 2 to 4 microns were present. This material, it will be recalled, contains not only asbestos fiber, but a considerable amount of iron and other material. Another series received a like amount of pure asbestos *fiber* cut and ground into the finest possible state. This fiber was obtained from a vein of Thetford ore. As a control, a third series was injected with a suspension of granite from Lorain County, Ohio. This rock contains free silica and ferrous carbonate, among other constituents. Samples of the peritoneal fluid were withdrawn with capillary pipettes twenty-eight and sixty-four days after the injections. In each series the fluid exhibited large numbers of dust containing monocytes, and in both the asbestos groups large giant cells were numerous. No sign of an asbestosis body could be discovered. Dissolved iron was detected in the cells from the granite series, but none could be found in those from either of the asbestos groups.

On the seventieth day and again on the 110th day, one animal from each group was killed. The amount of reaction was surprisingly insignificant. Careful search revealed minute foci of pigmentation in the omentum of each animal. At seventy days no suggestion of an asbestosis body was discovered, but at 110 days a few very small but typical bodies were found in a smear stained for iron of an omental nodule from one animal injected with the King's floats dust. The asbestos fiber and the iron containing granite animals did not show them. These findings were confirmed subsequently by histologic examination of the tissue. While the remaining animals of this experiment are still under observation, it hardly seems probable that asbestosis bodies will develop in any number. Aside from its failure to favor the formation of these structures, it is surprising that the peritoneal cavity exhibits so little reaction to the injection of very considerable quantities of this foreign material. When more insoluble dusts, such as coal, quartz, and carborundum, are injected, very appreciable deposits are found in the

omentum and beneath the parietal peritoneum.

In the subcutaneous tissues, attempts to form asbestosis bodies have been somewhat more successful. In an uncompleted experiment designed to determine whether certain dusts might cause a dissemination of infection with tubercle bacilli of attenuated virulence, daily repeated injections of asbestos dust suspensions were made into the subcutaneous tissue of the groin of guinea-pigs previously injected with tubercle bacilli in the same area. In all, thirty injections totaling 3 mg. of very fine dust (King's floats) were made. Most of the material has not yet been examined, but in one animal dying in 102 days, and in three more killed 264 days after the dust injections were completed, the subcutaneous tissues exhibited large masses of foreign body giant cells containing many typical asbestosis bodies.

With a view to determining whether the iron of the asbestosis body is derived from the dust or from the animal body, the following experiment has been performed. A mixture was prepared which contained equal parts of very finely divided pure asbestos fiber and fine whole asbestos dust (King's floats), the latter composed of particles between 2 and 4 microns in diameter. Both of these elements were included because the pure fiber from asbestos veins contains so little iron. The mixture was divided into two parts, and from one of them the iron was completely removed by digestion with hydrochloric acid and subsequent washing. Both fractions were sterilized by heating at 100°C. for one hour, and suspended in physiologic salt solution. The iron free portion formed a smooth turbid opalescent suspension, while the untreated portion produced a gray-green flocculent one. Two c.c. of each of these suspensions were injected into the subcutaneous tissues of the groins of each of six guinea-pigs; the untreated suspension on the right side, and the iron free material on the left. When examined thirty days later, all the animals had palpable nodules at the site of injection of the untreated material, and none on the opposite side. At this time one of the guinea-pigs was killed. On the right side there was a nodule 10 mm. in diameter filled with greenish-gray necrotic material, while on the left side there was only a faint suggestion of pigmentation in a very slightly enlarged superficial inguinal lymph node. Smears made from the material on each side failed to demonstrate golden-yellow bodies. When stained for iron with potassium ferrocyanide, numerous uniformly blue fibers were seen in the abscess produced by the untreated material, but only a few short, unstained fibers could be discovered in the smears from the opposite side. There was no suggestion of an asbestosis body. As in the case of the peritoneal cavity, the impression is gained that a part of the dust may have been dissolved in the body fluids. The effects produced at sixty days were identical. The absence of asbestosis bodies on the right side is attributable to the lack of sufficient time for their development; the disappearance of the injected material on the opposite side is most surprising.

(To be concluded)

BOOK REVIEWS

INDUSTRIAL HYGIENE FOR SCHOOLS. By *Jesse Feiring Williams*, M.D., Professor of Physical Education, Teachers College, Columbia University, and *Delbert Oberteuffer*, Ph.D., Supervisor of Health and Physical Education, Department of Education, State of Ohio. First edition. Cloth. Pp. xiv, 280 with index and illustrations. New York and London: McGraw-Hill Book Co., Inc., 1930.

This book gives a surprisingly detailed statement in regard to industrial health, together with many data upon personal hygiene. It should be suitable for high school students and for nurses. The latter will find it particularly useful as a guide to the educational work they are in a position to do. The book is attractively printed and thoroughly illustrated, and can be recommended without reserve to those desirous of an elementary text in industrial hygiene.—*C. K. Drinker.*

PERSONAL AND COMMUNITY HEALTH. By *Clair Elsmere Turner*, M.A., Dr.P.H., Professor of Biology and Public Health in the Massachusetts Institute of Technology; Formerly Associate Professor of Hygiene in the Tufts College Medical and Dental Schools; Sometime Member of the Administrative Board in the School of Public Health of Harvard University and the Massachusetts Institute of Technology; Fellow American Public Health Association; Major, Sanitary Corps, U. S. A. (Reserve). Cloth. Pp. 443 with index and illustrations. Third edition. St. Louis: The C. V. Mosby Co., 1930.

This is a thoroughly readable book on general hygiene, elementary in type and lacking in technical reference quality. It is well prepared and should be of use to nurses, safety men, and other persons without specific medical or public health training who are compelled to interest themselves in health work as pupils or administrators.—*Cecil K. Drinker.*

A TEXTBOOK OF HYGIENE. By *J. R. Currie*, M. A. Oxon., M. D. Glas., D. P. H. Birm., M. A., M. R. C. P. Edin., Professor of Public Health in the University of Glasgow; Examiner in Public Health, University of Aberdeen; Examiner for the Diploma in Public Health of the Royal Colleges of Physicians and Surgeons, Edinburgh, and the Royal Faculty of Physicians and Surgeons, Glasgow; Examiner to the Royal Sanitary Association of Scotland; Sometime Professor of Preventive Medicine, Queen's University, Kingston, Ontario; Medical Officer, Scottish Board of Health, and Medical Officer of Health of the County of Fife and the City of Chester. Cloth. Pp. 844 with index and illustrations. New York: Wm. Wood & Co.; Edinburgh: E. &. S. Livingstone, 1930.

This book is a technical summary of the practice of Hygiene in the British Isles. In our opinion it would be of little use elsewhere unless students desire to compare British practice with that of other countries. The presentation is in the form of a dispassionate listing of fact after fact and is not calculated to arouse interest in preventive medicine and hygiene as subjects intellectually attractive to the best medical students. In the United States, certainly, a realization has grown during the past ten years that the person-

nel entering Hygiene from our better medical schools will not be improved unless the subject can be given an intellectual status as stimulating as that of Medicine and Surgery. Dr. Currie's book covers the subject with photographic care but does not leave the reader with the inspiration which the subject deserves.—*Cecil K. Drinker.*

PROTOZOAN PARASITISM OF THE ALIMENTARY TRACT. Pathology, Diagnosis and Treatment. By *Kenneth M. Lynch*, M.D., Professor of Pathology, Medical College of the State of South Carolina, Charleston, South Carolina. Cloth. Pp. xvii, 258 with illustrations, references, and index. New York: The Macmillan Co., 1930.

Lynch has attempted to fill a long-felt need for an adequate compilation of the literature concerning the correlation of the finding of intestinal protozoa and certain symptom-complexes. The book is not intended to serve as another textbook of protozoology, of which there are already a sufficient number, of varying degrees of excellence; nor is it planned to be a complete system of parasitic diseases of the alimentary tract, giving comprehensive accounts of the symptomatology, pathology, differential diagnosis, prevention, and treatment of conditions known to be, or suspected to be, due to protozoan parasites. The book is calculated to aid the protozoologist and the practitioner to arrive at an understanding of the conditions likely to be met with, to enable them, either separately or together, to make a correct diagnosis and to give such cases rational treatment.

The book, taken as a whole, shows evidence of careful and painstaking work, and is well calculated to serve the purpose the author has in view. The subject matter is, except on a few minor points, treated with admirable conservatism. The manuscript appears to have been prepared two or three years ago or may have been unduly long in the hands of the publishers, for one finds but few references, even to the more important papers which have appeared since 1927. Reference is made to papers such as that of Hegner and Ratcliffe (*Jour. Parasitol.*, 1927–1928, *14*, 27) dealing with the trichomonads of the vagina of the monkey, but important work, such as that of Dobell (*Parasitol.*, 1928, *20*, 357) and of Cleveland and Sanders (*Arch. f. Protistenk.*, 1930, *70*, 223), dealing with such fundamentals as the life history of *Entamoeba histolytica*, is ignored. Omissions of this nature are not infrequent.

Contrary to the practice of most competent protozoologists, Lynch consistently uses *Endamoeba* instead of *Entamoeba* as the generic name for the commoner and more important intestinal amoebae of man. He gives no reasons for doing so. The statement is made (p. 31) that "all but one of the species (amoebae) found in man are known to undergo encystment." He correctly indicates that *Entamoeba gingivalis* does not encyst, but later (p. 123) states that the cysts of *Dientamoeba fragilis* are relatively unknown. A cyst of *Dientamoeba fragilis*, hitherto described only by Kofoid, is figured on page 122, but no description is given of this interesting body, whose very existence is problematical. Too much space is devoted to a consideration of the existence or nonexistence of *Councilmania lafleuri*, *Karyamoebina falcata*, and *Caudamoeba sinensis*. Similarly, *Entamoeba gingivalis* and

Trichomonas buccalis are given undue prominence in the discussion of the etiology of pyorrhea alveolaris, and the author's treatment of this subject is not calculated to clear up the confusion.

Several statements made by the author would lead the inexperienced and unwary reader to believe that the trichomonads present considerable difficulty to cultivation on artificial media *in vitro*.—*Hans Theiler.*

BOOKS RECEIVED

Books received are acknowledged in this column, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers and as space permits.

PERSONAL HYGIENE FOR NURSES. By *Seneca Egbert*, A.M., M.D., Dr. P. H., Professor of Hygiene, University of Pennsylvania; Formerly Professor of Hygiene and Dean of the Medico-Chirurgical College of Philadelphia, and Professor of Anatomy and Physiology in Temple University; Lecturer at Schools of Nursing of the Graduate Hospital of the University of Pennsylvania and the Philadelphia General Hospital. Cloth. Pp. 347 with index and illustrations. Philadelphia: F. A. Davis Co., 1930.

TREATMENT OF EPILEPSY. By *Fritz B. Talbot*, M.D., Clinical Professor of Pediatrics, Harvard University Medical School; Chief of Children's Medical Department, Massachusetts General Hospital. Cloth. Pp. 308 with bibliography and index. New York: The Macmillan Co., 1930.

A MANUAL OF THE COMMON CONTAGIOUS DISEASES. By *Philip Moen Stimson*, A.B., M.D., Associate in Pediatrics, Cornell University Medical College; Attending Physician, Willard Parker Hospital; Chief of Staff, the Floating Hospital of St. John's Guild; Chief of Clinic, Department of Pediatrics, Cornell Clinic; School Physician, the Horace Mann Schools, 1919–1923; President, the School Physicians Association, 1928–1930. Fabrikoid. Pp. xvi, 353, with index and illustrations. Philadelphia: Lea & Febiger, 1931.